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The Role of Serum IL-10 And IL-17 Levels in The Progression and Severity of Bacterial Pneumonia

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Abstract

Background: Bacterial pneumonia is still a leading cause of global morbidity and mortality. Interleukin-10(IL-10) and interleukin-17(IL-17), two major cytokines participating in the anti-inflammatory and pro-inflammatory pathways. The objective of the study was to determine whether serum IL-10 and IL-17 acts as a noninvasive marker for bacterial pneumonia staging and severity. **Methods**: A case-control study was carried out at Al-Sadr Medical City, Al-Najaf, Iraq. This study enrolled 60 patients diagnosed as bacterial pneumonia (cases) and 40 apparently healthy controls. Serum IL-10 and IL-17 using Enzyme-linked immunosorbent assays (ELISA). According to the severity of illness, patients were divided into groups with mild, moderate and severe pneumonia. **Results**: Serum IL-10 and IL-17 levels in bacterial pneumonia patients were also significantly elevated when compared with healthy controls ($P < 0.001$). The average IL-10 level was 32.84 ± 8.71 pg/mL in patients vs. 12.63 ± 4.25 pg/mL in controls and the average IL-17 level was 48.57 ± 11.36 pg/mL vs. 18.92 ± 6.14 pg/mL, respectively. Moreover, both cytokines increased significantly with disease severity. Among the three groups, patients with severe pneumonia had the highest concentrations of IL-10 and IL-17 ($P = 0.002$ and $P < 0.001$, respectively). **Conclusions**: High serum IL-10 and IL-17 levels associated with bacterial pneumonia can be used as a significant correlate with severity of disease. Therefore, these results indicate that both cytokines should be helpful for predicting or evaluating the disease severity and disease course of patients with bacterial pneumonia.

Keywords: IL-10, IL-17, Pneumonia, Streptococcus pneumoniae, Haemophilus influenzae, Mycoplasma pneumoniae

Introduction

Despite remarkable advances in antibiotic therapy and prevention strategies, bacterial pneumonia continues to be one of the most common causes of morbidity and mortality worldwide. It is defined by an acute inflammatory response of the lung parenchyma triggered by bacterial invasion, which evokes a

complex host immune response that involves both innate and adaptive components (Sattar et al., 2026). The clinical outcomes of bacterial pneumonia depends not only on the characteristics like virulence of the infecting pathogen but also on the intensity and regulation of the host inflammatory response. It is also too much or intolerable amount of cytokines produced in a short time, which may lead to tissue damage, respiratory failure and bad clinical outcome (Feng et al., 2021).

Cytokines are important mediators of immune response in pulmonary infections. IL-17 and IL-10 have particularly garnered attention from immunologists due to their antagonistic immune roles. IL-17 is a pro-inflamatory cytokine mainly produced by T helper 17 (Th17) cells, and other immune cells. It stimulates neutrophil recruitment, increased chemokine and inflammatory mediator production and antimicrobial defense against extracellular bacterial and parasitic pathogens (Hadi et al., 2022). These functions are crucial for the clearance of bacteria, but elevated IL-17 production may worsen pulmonary inflammation and lead to lung injury (Hadi et al., 2024).

Recent data has underscored the clinical importance of IL-17 in pneumonia. In a prognostic cohort of patients with community-acquired pneumonia (CAP), a prospective study showed that serum IL-17 concentrations correlate with the severity of CAP, as well as poor prognostic outcome with ICU admission, mechanical ventilation, hospitalization duration and mortality. These results imply the potential role of IL-17 as a biomarker to predict disease severity and prognosis in patients with pneumonia, (Feng et al., 2021). In addition, increased IL-17 are associated with prolonged inflammatory response in diverse pulmonary disorders, further supporting the role of IL-17 as mediator of tissue inflammation and disease progression (Ritzmann et al., 2022).

On the other hand, IL-10 is a powerful anti-inflammatory cytokine that is needed for maintaining immune homeostasis. IL-10 is secreted by regulatory T cells, macrophages and other immune cells and suppresses pro-inflammatory cytokines production and excessive immune activation. IL-10 serves as a brake against excessive inflammation and tissue injury associated with bacterial pneumonia. However, high levels of IL-10 production may also retard clearance rather than promote it through down-regulation of protective immune responses, leading to persistent infection and poor clinical outcomes (Morrison et al., 2000).

Finding the appropriate balance of proinflammatory and anti-inflammatory cytokines seems to be key in determining severity and ultimate clinical course of pneumonia. Others showed that the noisy ratio and crosstalk of inflammatory mediators can differentiate between mild and severe forms of pulmonary infection. The balance between IL-6 and IL-10 differentiates severe acute pneumonia from milder disease, reflecting the adverse influence of uncontrolled cytokine production which helps to explain the pathogenesis of disease (Brito et al., 2016). This suggests that evaluating IL-17 along with IL-10 would give a context for the immune dynamics in bacterial pneumonia.

Recent advancements in immunology have refocused attention to the question of determining reliable biomarkers that are predictive of disease severity, can track changes over time and may indicate response to treatment. Cytokine profiling has recently been outlined as an attractive alternative since it potentially reproduces more faithfully the biological pathways that are in play at the time of infection when compared to standard inflammatory markers alone. However, the function of IL-10 and IL-17 in the development of bacterial pneumonia is not well defined. In conclusion, the investigation of these cytokines association with severity of the disease may help us to understand the host-pathogen relationships that could manage strategies in order to develop new diagnostic and prognostic purposes.

Accordingly, the goal of this study is to assess serum IL-10 and IL-17 levels in patients with bacterial pneumonia and analyze their correlation with disease progression and severity. The relationship between these cytokines could be useful to the understanding of mechanisms that mediate the immunopathogenesis of bacterial pneumonia and may help to identify new biomarkers for clinical use.

Methods

Study Design

A case-control study including 160 patients with bacterial pneumonia was performed at Al-Sadr Medical City, Al-Najaf, Iraq during the period from April to November 2025 to assess the role of serum interleukin-10 (IL-10) and interleukin-17 (IL-17) levels in progression and severity of bacterial pneumonia.

Participants

This non-randomized, controlled study included 100 participants: 60 patients with bacterial pneumonia diagnosed according to clinical examination and historical data; and 40 apparently healthy individuals (the control group). Patients were recruited through the Respiratory Medicine Department and inpatient wards of Al-Sadr Medical City. Specialist physicians made the diagnosis of bacterial pneumonia based on clinical

manifestations, radiological findings and microbiological investigations.

Eligible patients were defined as adult (age ≥ 18 years) individuals presenting with signs and symptoms consistent with bacterial pneumonia, which included fever ≥ 100.4°F (38°C), productive cough, dyspnea, chest pain and radiographic evidence of pulmonary infiltrates. Also excluded were patients with viral pneumonia, tuberculosis, chronic inflammatory diseases, autoimmune disorders or malignancies—these are factors that plant immunity has been found to impair—or those receiving immunosuppressive therapy.

The control group was composed of healthy volunteers, as similar in age and sex as possible. At the time of sample collection, controls had no clinical evidence of respiratory infection, chronic inflammatory disease or any other major medical condition.

Bacterial Diagnosis

Through clinical picture, radiological finding, laboratory test, and microbiological test, a diagnosis of bacterial pneumonia was made. Specialist physicians evaluated patients whereby diagnosis was established either by clinical symptoms compatible with pneumonia (fever, cough, dyspnea, chest pain) with pulmonary infiltrates evident from chest radiographs. For all patients, respiratory specimens (sputum) were collected prior to the initiation of antibiotic therapy whenever feasible. The samples were immediately delivered to the microbiology laboratory for processing and bacteriological analysis. Specimens were continuously cultured on suitable selective and enriched media as well as incubated under suitable conditions, as per standard microbiological procedures. Identification of bacterial isolates was largely based on a variety of factors: colony morphology, Gram-staining characteristics and biochemical reactions Confirmatory tests as necessary according to OIE guidelines were also performed to aid species identification. The microbiological study of patients with pneumonia identified such bacteria as key pathogens in its causation as: *Haemophilus influenzae, Streptococcus pneumoniae, Mycoplasma pneumoniae*. Using conventional microbiological methods and species-specific laboratory tests where applicable, the identification of these organisms was performed.

Blood Sample Collection

Peripheral blood (5 ml) was collected from all subjects using sterile disposable syringes. Samples were then placed in plain tubes and allowed to clot at room temperature. Serum was separated by centrifuging the bloods at 3000 rpm for 10 min and kept in sterile Eppendorf tubes at −20°C until analyzed.

Measurement of serum IL-10 and IL-17

Concentrations of IL-10 and IL-17 in serum were measured using commercially available enzyme-linked immunosorbent assay (ELISA) kits (HumaCount, Germany), which were performed according to the manufacturer's instructions. In short, standards and sera were added to the wells of a microplate which had been coated with monoclonal antibodies. Detection antibodies conjugated with an enzyme were added along with chromogenic substrates following a series of incubation and washing steps. Using an ELISA microplate reader, the optical density was evaluated at 450 nm and cytokine concentrations were calculated from standard calibration curves. All samples were analyzed in duplicate to ensure the accuracy and reproducibility of the experiments.

Assessment of Disease Severity

Clinical and laboratory parameters were used to determine the severity of bacterial pneumonia at the admission: body temperature, respiratory rate, oxygen saturation (Sp9o2), total white blood cell count, routine chest x-ray and radiological extent image-lung affection. Comparison analyses were conducted by stratifying patients based on disease severity.

Ethical Considerations

Ethics approval and consent to participateThe study protocol was reviewed and approved by the Research Ethics Committee of Al-Sadr Medical City, Al-Najaf, Iraq. All the subjects gave informed consent before enrollment. Participant response privacy (e.g., confidentiality and anonymity).

Statistical Analysis

Statistical analyses were performed in IBM SPSS Statistics version 26. Continuous outcomes were presented as mean ± standard deviation (SD) while categorical variables will be summarized by frequencies and percentages. The independent-samples t-test was used for continuous variables, and the chi-square test for categorical variables to compare groups. Correlations between serum IL-10 and IL-17 levels with different severity parameters were assessed by Pearson's correlation. Point-wise significance was defined as $p < 0.05$. ANOVA Test and least significant difference (LSD) method were utilized to determine the differences in serum IL-10 and IL-17 of patients subgroups categorized according to the disease severity (Al-fahham, 2018).

Results

Table 1 demonstrates the distribution of age, sex, residence, and smoking status among patients with bacterial pneumonia and healthy controls. The age distribution was comparable between the two groups, with the highest proportions observed in the 38–47-year age category among patients and the 28–37-year age category among controls. No statistically significant difference was detected regarding age distribution (P = 0.69). Likewise, males constituted a higher proportion of participants in both groups; however, the difference was not statistically significant (P = 0.28). Urban residents were more common among patients than controls, although this difference did not reach statistical significance (P = 0.06). Smoking was more frequently reported among patients compared with healthy controls, but the association was also non-significant (P = 0.06).

Table 1. Age, sex, residence and smoking distribution of patients with pneumonia and healthy control

Items		`Patients (No. = 60)		Control (No. = 40)		Chi Square	P value (Sig.)
		Freq.	%	Freq.	%		
Age/Years	18-27	12	20	10.00	25	1.42	0.69 (NS)
	28-37	16	26.7	11	27.5		
	38-47	17	28.3	9	22.5		
	≥ 48	15	25	10	25		
Sex	Male	37	61.7	21	52.5	1.15	0.28 (NS)
	Female	23	38.3	19	47.5		
Residence	Urban	39	65	20	50	3.54	0.06 (NS)
	Rural	21	35	20	50		
Smoking	Yes	24	40	10	25	2.67	0.10 (NS)
	No	36	60	30	75		

NS: Non-significant at P>0.05

Among pneumonia patients diagnosed with bacterial pathogens, *Streptococcus pneumoniae* is the most prevalent at 48.5%, followed by *Haemophilus influenzae* at 28.4%, and *Mycoplasma pneumoniae* at 23.1% (Figure 1).

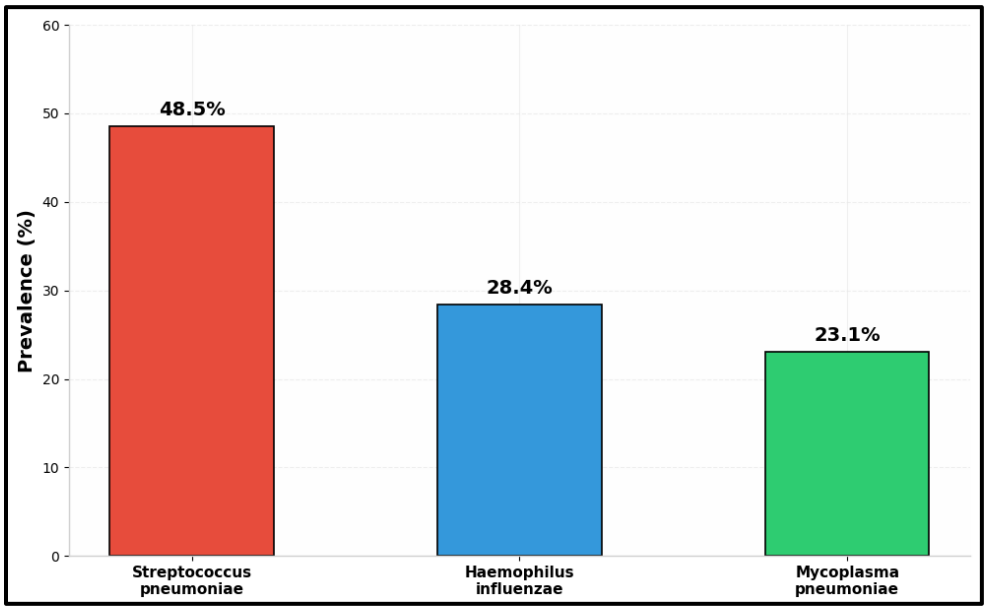

Figure 1. Distribution of patients according to bacterial causative species

The bar chart in figure 2., shows that the majority of patients were diagnosed with moderate severity 51.7%, followed by mild pneumonia at 32.4%, while the sever cases was reported only in 14.9% (Figure 2).

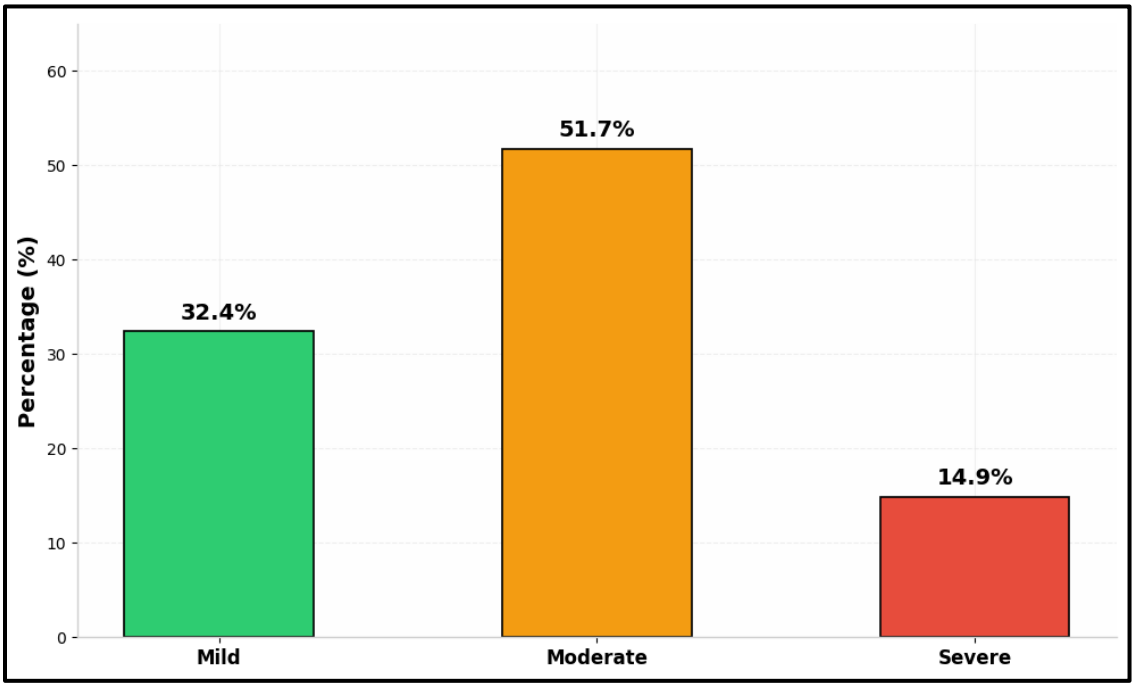

Figure 2. Distribution of patients according to the severity of the pneumonia

Comparison of serum IL-10 and IL-17 concentrations between patients with bacterial pneumonia and healthy controls (Table 2). Patients had a higher mean serum IL-10 concentration (32.84 ± 8.71 pg/mL) compared with controls (12.63 ± 4.25 pg/mL), and this difference was statistically significant ($P < 0.001$). Correspondingly, serum IL-17 levels of pneumonia patients (48.57 ± 11.36 pg/mL) also were markedly higher than in the control group (18.92 ± 6.14 pg/mL), with a highly significant difference between both groups again ($P < 0.001$).

Table 2. Assessment of IL-10 and IL-17 levels between patients with pneumonia and control subjects

Markers	Patients (n=60) (Mean ± S.D)	Control (n=40) (Mean ± S.D)	T Test (P Value)
IL-10 (pg/ml)	32.84 ± 8.71	12.63 ± 4.25	P < 0.000 (HS)
IL-17 (pg/ml)	48.57 ± 11.36	18.92 ± 6.14	P < 0.000 (HS)

HS: High significant at P<0.001

Serum levels of IL-10 according to severity of bacterial pneumonia is shown in Table 3. The mean serum IL10 concentration progressively increasing along with disease severity from 27.42 ± 5.83 pg/mL in mild pneumonia to 35.76 ± 7.14 pg/mL in moderate patients and finally reached a level of (46.88 ± 8.96) pg/mL among patients with severe pneumonia. The results of the statistical analysis showed a very significant difference between the three severity groups ($F = 6.84$, $P = 0.002$).

Table 3. Differences in IL-10 levels in patients' groups according to the severity od pneumonia

Disease Severity	Freq.	IL-10 (pg/ml) Mean ± S.D	F test	T test P-value
Mild	31	27.42 ± 5.83	6.84	0.002 (HS)
Moderate	20	35.76 ± 7.14		
Severe	9	46.88 ± 8.96		

HS: High significant at P<0.01

Serum IL-17 levels measured among groups formed according to severity of bacterial pneumonia are shown in Table 4. A gradual rise in levels of IL-17 has been observed; it is correlated with increased disease severity. The mean IL-17 level was significantly lower in patients with mild pneumonia (39.84 ± 8.27 pg/mL) than those with moderate and severe pneumonia (52.31 ± 9.65 pg/mL, $P < 0.05$ and 67.72 ± 11.48 pg/mL, $P < 0.01$ respectively). From the statistical analysis, there was a highly statistically significant difference between the three severity groups ($F = 11.27$, $P < 0.001$).

Table 3. Differences in IL-17 levels in patients' groups according to the severity of pneumonia

Disease Severity	Freq.	IL-17 (pg/ml) Mean ± S.D	F test	T test P-value
Mild	31	39.84 ± 8.27	11.27	<0.001 (HS)
Moderate	20	52.31 ± 9.65		
Severe	9	67.72 ± 11.48		

HS: High significant at P<0.01

Discussion

The current study was performed to assess the relationship of serum levels of interleukin-10 (IL-10) and interleukin-17 (IL-17) with the progression and severity of bacterial pneumonia. These results revealed markedly higher levels of these two cytokines in subjects who had pneumonia compared to healthy controls. In addition, serum IL-10 and IL-17 levels were significantly increased in patients with bacterial pneumonia in a progressive manner with increasing severity of pneumonia, highlighting the potential role of these cytokines in the immunopathogenesis of bacterial pneumonia.

Patients and controls did not differ significantly in terms of age, sex, residence, and smoking status. The comparability of study groups between UA vs non-UA patients reflects a reduction in the possible effect of factors that could confound such associations, and thereby the validity of the observed associations between cytokine levels and disease status. Interestingly, studies focused on inflammation research within infectious diseases settings have recommended such demographic matching which could set up for such prospective evaluations, with the aim of showing that the majority of immunological alterations observed were disease processes related (Torres et al., 2021).

An important finding of the present study was the significantly higher serum levels of IL-10 in patients with bacterial pneumonia compared to healthy controls. Interleukin-10 (IL-10) is an anti-inflammatory cytokine that are secreted by regulatory T (Treg) cells, macrophages, dendritic cells, and B lymphocytes. The primary purpose is to inhibit inflammatory reactions that are hyperactive, and prevent tissue damage during an infection. The higher levels of IL-10 observed in the present study might represent a homeostatic mechanism, which is directed toward the regulation of the excess in the inflammatory reaction evoked by bacterial pathogens in the lung. These results were also reported by van der Poll and Opal (2008) who showed that high concentrations of IL-10 have been reported in patients with severe bacterial infections where the immunoregulatory effects of IL-10 on the host immune response are critical. In addition, Restrepo et al. (2008) reported significantly increased circulating IL-10 levels among patients with community-acquired pneumonia, particularly in those with severe disease manifestations.

The current analysis further showed a strong correlation between plasma levels of IL-10 and the severity of disease. The highest levels of IL-10 were observed in severe pneumonia patients, followed by moderate and then mild disease. These data suggest that increased IL-10 production occurs when inflammatory burden increases. IL-10, which inhibits host tissues against inflammatory injury, but its overexpression can inhibit protective immune mechanisms and reduce bacterial clearance. Previous studies referring to this dual role also indicate an association

between higher IL-10 levels, and higher disease severity, longer hospitalization, and, eventually, a worse clinical outcome. IL-10 therefore may play a role in not only reflecting immune regulation but may also predict the outcome of bacterial pneumonia (Kumar et al., 2009).

Another significant finding is the high level of serum IL-17of pneumonia cases compared with controls. Interleukin 17 (IL-17) is a pro-inflammatory cytokine, mainly produced by a subset of CD4 + T cells known as Th17 cells, and plays a central role in host defense against extracellular bacterial pathogens. IL-17 induces the recruitment of neutrophils, as well as the production of chemokines and antimicrobial peptides at sites of infection. High concentrations of IL-17 in the present study are thus consistent with the biological function of this cytokine in lung innate immunity against bacteria. In this regard, prior studies have shown the induction of IL-17 in patients with bacterial respiratory infections (Ye et al., 2001; Li et al., 2023).

This experimental contribution of IL-17 to in vivo bacterial clearance in lungs is thus in line with the current findings. Ye et al. IL-17 mediates the mobilization of neutrophils and has a potent role in host defense against pulmonary bacterial diseases (2001). Additionally, Li et al. (2023) characterized increased IL-17 levels as an independent correlate of inflammatory activity and disease progression in patients with severe bacterial pneumonia. These findings suggest that IL-17 is an orchestrator of both innate and adaptive immune paths in the lung upon pulmonary infection. An In addition, serum IL-17 concentrations were markedly increased according to pneumonia severity. Acting on IL-17 in patients with severe pneumonia they found significantly higher levels of IL-17 in these patients in comparison with patients with moderate and mild disease. This pattern suggests a higher association between Th17 activation and disease progression and greater inflammatory burden. IL-17-mediated production in moderate concentrations is beneficial in clearing the pathogens, but high release of IL-17 might be implicated in the pulmonary tissues damage, injury of alveoli, and could promote the progression of respiratory failure. To date, comparable findings have only been detailed in regards to serious respiratory attacks, where supranormal IL-17-influenced irritation was associatedwith lowered clinical productivity (Crowe & Chen, 2020).

The increase in IL-10 and IL-17 between individuals in the present study implies that there are complex relationships between pro- and anti-inflammatory pathways during bacterial pneumonia. IL-17 is proinflammatory and aids in bacterial clearance, and IL-10 acts to curtail excessive immune activation and tissue damage. This apparent balance of opposing immune regulators may be important in terms of disease progression and clinical outcome. Imbalance during this process results either in rampant bacterial infection due to immune inhibition or gigantic tissue damage due to excessive inflammation. Analogous immunological interactions were reported in cytokine networks during severe pulmonary infections and sepsis (van der Poll & Opal, 2008).

The significant increases of IL-10 and IL-17 in pneumonia patients and the correlation with the severity of the disease make both cytokines good candidates to be used as biomarkers for monitoring and prognosis of the disease. These cytokines measurement may facilitate recognition of patients at elevated risk of severe disease, and may permit improved therapeutic decision-making. Additionally, the results have implications for the ongoing exploration of therapeutic strategies targeting cytokines to dampen host responses that are deleterious but that do not impair effective antibacterial immunity.

Conclusion

The present study revealed that the serum concentration levels of both IL-10 and IL-17 in patients with bacterial pneumonia was significantly higher compared to healthy control subjects, and they gradually increased according to disease severity. Against bacterial pneumonia, this emphasizes the critical role of both effector and suppressive anti-inflammatory immune responses during disease development. This observed associations provide evidences that IL-10 and IL-17 may be used as potential biomarkers of disease progression and severity. The prognostic implications of IL-10 and IL-17 derived scores, as well as their potential role as targets of therapeutic intervention in bacterial pneumonia deserve further investigation in larger studies with extended follow-up.

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